11/00 KAL

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     FILE 'MEDLINE, BIOSIS' ENTERED AT 15:51:16 ON 26 NOV 2000
L1        605566 S OSTEOCLAST# OR OSTEOBLAST# OR BONE
L2         65551 S ((INTERFERON OR INF)(A)(B OR G OR BETA OR GAMMA)) OR (INFB
OR
L3          2744 S L1 AND L2
L4          1241 S L3 AND (CANCER OR TUMOR OR MYELOMA OR METASTA? OR
CARCINOMA#)
L5           154 S L3 AND TUMOUR
L6          1280 S L4 OR L5
L7           808 S L6 AND HUMAN
L8           504 S L7 AND PY<1996
L9        217888 S L1/TI
L10          823 S L9 AND L2
L11          340 S L10 AND (CANCER OR TUMOR OR TUMOUR OR MYELOMA OR METASTA?
OR
L12          236 DUP REM L11 (104 DUPLICATES REMOVED)
L13          138 S L12 AND PY<1996
L14        14007 S OSTEOCLAST#
L15       265428 S METASTATIC OR METASTASIS
L16          538 S L15 AND L14
L17         3427 S L14(S)(INCREASE OR ACTIVATION OR UPREGULATION OR
(UP-REGULATI
L18          126 S L17 AND L15
L19           89 DUP REM L18 (37 DUPLICATES REMOVED)
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Related Articles

TITLE: Nitric oxide and bone.

AUTHORS: Evans DM; Ralston SH

AUTHOR AFFILIATION: Department of Medicine and Therapeutics, University of Aberdeen, Scotland, UK.

SOURCE: J Bone Miner Res 1996 Mar;11(3):300-5

CITATION IDS: PMID: 8852940 UI: 97005641

ABSTRACT: Nitric oxide (NO), a mediator of cardiovascular homeostasis, neurotransmission, and immune function, has recently been found to have important effects in bone. Both constitutive and inducible forms of NO synthase are expressed by bone-derived cells, and cytokines such as interleukin-1 (IL-1), tumor necrosis factor (TNF), and interferon gamma (IFN-gamma), are potent stimulators of NO production. When combined with other cytokines, IFN-gamma markedly induces NO production, which suppresses osteoclast formation and activity of mature osteoclasts. This "superinduction" of NO is largely responsible for the selective inhibitory effect of IFN-gamma on cytokine-induced bone resorption. High concentrations of NO are also inhibitory for cells of the osteoblast lineage, and NO production appears to be partly responsible for the inhibitory effects of cytokines on osteoblast proliferation. At lower concentrations, however, NO has different effects. Moderate induction of NO potentiates bone resorption, and the constitutive production of NO at low concentrations promotes the proliferation of osteoblast-like cells and modulates osteoblast function. NO therefore appears to be an important regulatory molecule in bone with effects on cells of the osteoblast and osteoclast lineage and represents one of the molecules produced by osteoblasts which directly regulate osteoclastic activity. Stimulation of NO production in bone by proinflammatory cytokines raises the possibility that NO may be involved as a mediator of bone disease in conditions associated with cytokine activation, such as rheumatoid arthritis, tumor associated osteolysis, and postmenopausal

	osteoporosis.
MAIN MESH HEADINGS:	Bone and Bones/*metabolism Nitric Oxide/*biosynthesis
ADDITIONAL MESH HEADINGS:	Arthritis, Rheumatoid Bone Resorption/chemically induced Enzyme Induction/drug effects Female Free Radicals Human Interferon Type II/metabolism Interferon Type II/pharmacology Interleukin-1/metabolism Interleukin-1/pharmacology Nitric Oxide/metabolism Nitric-Oxide Synthase/metabolism Osteoblasts/cytology Osteoblasts/metabolism Osteoclasts/cytology Osteoclasts/metabolism Osteoporosis, Postmenopausal/etiology Support, Non-U.S. Gov't Tumor Necrosis Factor/metabolism Tumor Necrosis Factor/pharmacology 1996/03 1996/01 00:00
PUBLICATION TYPES:	JOURNAL ARTICLE REVIEW REVIEW, TUTORIAL
CAS REGISTRY NUMBERS:	EC 1.14.13.39 (Nitric-Oxide Synthase) 0 (Free Radicals) 0 (Interleukin-1) 0 (Tumor Necrosis Factor) 10102-43-9 (Nitric Oxide) 82115-62-6 (Interferon Type II)
LANGUAGES:	Eng

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